Clostridium botulinum type C intoxication in feedlot steers being fed ensiled poultry litter

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Three cross-bred, beef steers were presented to the Veterinary Teaching Hospital at the University of Montreal with a chief complaint of sudden generalized weakness and recumbency. Approximately 15 other steers with similar clinical signs had died during the past week on the same farm. Postmortem examination of 2 animals showed no macroscopic lesions. The animals were fed a ration consisting of 60% dry corn, 20% ensiled chicken litter, 13% whole potatoes, 3% timothy haylage, 3% prepared milk by-product, and 3.3 grams/head/d mineral supplement. On arrival at the feedlot, steers had been immunized against clostridial diseases and *Haemophilus somnus* (Fermicon 7 Somnugen, Boehringer Ingelheim (Canada) Ltd., Burlington, Ontario), respiratory diseases (Horizon-IV, Bayvet Concord, Ontario; TSV-2, SmithKline Beecham, Mississauga, Ontario), and gram-negative endotoxemia (Endovac, Bayvet, Concord, Ontario). The feedlot housed several hundreds of cross-bred animals ranging from 250 to 500 kg. Most affected animals were from a lot that housed 250 animals.

On admission, the 3 steers were in sternal recumbency and unable to stand, even if they were stimulated and given assistance. On physical examination, the absence of ruminal contractions was noticed. Neurological examination revealed a slight lack of tail tone, and tetraparesis was suspected. Tongue tone was assessed to be normal and oral examination was unremarkable. Two of the 3 animals were able to eat and drink normally. Vital signs as evaluated on arrival were within normal range (rectal temperature: 38.2°C to 38.7°C, heart rate: 60 to 80 beats/min, and respiratory rate: 20 to 24 breaths/min). The pupillary light response was diminished and a slight mydriasis was noticed in both eyes. The animals had a normal mental status. They passed dry feces, and micturition was judged to be normal.

Ancillary tests performed included a complete blood count (CBC), biochemical profile analysis (BPA), determination of ruminal pH, venous blood gas analysis, and cerebrospinal fluid (CSF) collection at the lumbosacral junction. The CBC revealed a neutrophilic leukocytosis in all 3 animals (WBC = 12.5 to 13.2 $\times$ 10^9 cells/L) and an increased plasma fibrinogen concentration (7 to 11 g/L). The BPA revealed elevation of aspartate aminotransferase (AST) (163 to 749 µ/L), creatine phosphokinase (CPK) (897 to 13 780 µ/L), and total CO_2 (31.3 to 36.0 mmol/L). The rumen pH ranged from 8 to 8.5. Blood glucose concentrations (4.2 to 5.6 mmol/L) and serum calcium concentrations (2.53 to 2.64 mmol/L) were within the normal range for all 3 animals. The venous blood gas analysis revealed a slight metabolic alkalosis. The cytological, biochemical, and bacteriological cultures of CSF were unremarkable.

Initial treatment (on arrival) included IV fluid therapy of isotonic saline solution, supplemented with calcium borogluconate (500 mL of a 23% solution in 20 L) at a rate of 4 mL/kg body weight/h. Treatment was instituted before the laboratory results were available; supplementation with calcium was discontinued when test results showed a normal serum calcium value (4 h postarrival). Vitamin E and selenium (Dystosel: 3 mg selenium and 136 IU of vitamin E/mL, rogar/STB, London, Ontario) (8 mL, IM, single dose) and 2 L of ruminal contents from a normal cow were also administered at that particular time.

Despite the treatment, 2 steers deteriorated in condition during the following 24 h. They developed laborious breathing without any specific pattern and were unable to stay in sternal recumbancy, so they were euthanized. Lesions were not found on postmortem examination. Bacteriological cultures of lungs and the central nervous system were negative. The 3rd steer remained stable for the next 5 d, although paresis seemed to be more pronounced in the forelimbs. Then his condition improved gradually during the following 7 d, at which point he was able to remain standing without external support. He was subsequently discharged.

The slight elevation in AST and CPK suggested muscle damage due to trauma and/or recumbency. This slight increase in muscle enzymes, combined with the absence of muscle lesions on histological examination, was not suggestive of the nutritional myodegeneration initially suspected. A metabolic disease was not likely, considering the fact that no electrolyte imbalance was indicated in the BPA. An infectious neurological condition was eliminated by the results of the CSF analyses. The combination of tetraparesis, normal CSF, and the absence of severe myopathy localized the problem to the peripheral nerves or the neuromuscular junctions, which, combined with the relatively large number of animals affected, led to a presumptive diagnosis of botulism. During this period, 5 steers from the same feedlot, presenting similar clinical signs, were submitted to 2 different federally-inspected slaughterhouses. Ruminal contents, feces, and sera were submitted for detection of *Clostridium botulinum* toxin. The Botulism Reference Service Laboratory of the Health Protection Branch of Health Canada in Ottawa detected the presence of type C toxin of *C. botulinum* in a pooled serum sample from 2 of the steers. Subsequently, the same laboratory was able to demonstrate the presence of viable cells of *C. botulinum* in individual rumen samples from these 2 steers. A diagnosis of intoxication caused by *C. botulinum* type C was made.

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In the clinical bacteriology laboratory of the Veterinary Teaching Hospital, 2 CF-1 mice (25 to 30 g) were each injected intraperitoneally (IP) with 1 mL of serum from 1 of the steers that subsequently died. These mice developed clinical signs (reluctance to move to complete incapacity to move, tachypnea, severe dyspnea, ruffling of the hair), followed rapidly by death (30 and 50 h postinjection). Another mouse was injected with the serum taken from another of the steers that subsequently died (1 mL, IP), as well as 1 mL of serum from the 3rd steer that ultimately survived. This mouse did not develop any clinical signs. Yet another mouse injected only with the serum from the 3rd steer did not develop clinical signs. This experiment suggested that serum from the 3rd steer did not contain active toxin but did contain antitoxin, which provided protection against the *C. botulinum* toxin.

During the following 2 wk, 32 other animals at the feedlot developed similar clinical signs. Typically, animals were found in the morning in sternal recumbency, refusing to get up, even if stimulated. They then deteriorated over anywhere from 24 to 72 h. Clinical signs were similar to those described for the animals presented to the hospital. Rare cases survived several days and were euthanized for humane reasons. Total mortality, excluding animals presented for slaughter, was 35. In the meantime, the suspected presence of botulinum toxin in the poultry litter had prompted the feedlot owner to withdraw the litter from the ration. No new cases were reported after the 10th d following removal of the litter.

Botulism in cattle and other mammals is caused by a neurotoxin produced by *C. botulinum*. The bacterium is a strictly anaerobic, spore-forming, gram-positive rod. Eight different types of botulinum toxins have been identified: types A, B, Ca, Cb, D, E, F, and G (1). Bovine botulism has been reported in Australia, South Africa, and the Gulf Coast area of the United States (2). Cattle can be affected by types B, C, and D. The disease in cattle in Europe, Australia, and South Africa seems to be more commonly associated with *C. botulinum* types C and D, while in North America type B seems to be more important. Recently, an important outbreak of botulism type C intoxication was reported in Australia (mortality rate was 8%, 31%, and 40% in 3 farms, where 1421 animals were exposed and 376 died) (3). The intoxication was related to the use of poultry litter applied as a fertilizer on pasture.

Poultry litter is used as a supplement in several countries, including Canada, Ireland, and other countries in Europe, as well as in South Africa and the United States. The use of ensiled poultry litter as a dietary supplement for feedlot cattle would seem to provide benefits for both poultrymen and cattlemen alike. One benefit is to be able to dispose of an unwanted substance, and the other is to provide a cheap source of protein. This practice does, however, present certain risks. Type C intoxication is most commonly reported in North America in avian species (4). Other sources of contamination exist in cattle and include brewer's grains (5), rye silage (6), and high moisture pit-ensiled corn (7). In this particular case, we believe the poultry litter was the origin of the toxin. However, according to the owner, no poultry carcasses were found in the litter at any time.

Typically, 3 types of botulism are reported: ingestion botulism, toxicoinfectious botulism, and wound botulism (1). Ingestion botulism involves the intake of preformed toxin and seems to be the most common type of botulism in animals (1). In most cases, the toxin penetrates the gastrointestinal mucosa and reaches the neuromuscular junction via the circulatory system. Three steps are necessary for the irreversible binding of the toxin to the motor end plate: 1) binding of the toxin to a receptor on the external surface of the nerve terminal, 2) translocation to a site within the nerve terminal, and 3) binding to an internal receptor site where the toxin blocks the release of the neurotransmitter acetylcholine (4).

The typical clinical signs of bovine botulism usually include dysphagia, generalized muscular weakness, reduced tongue tone, and bradycardia (30% of cases) (4). Affected animals are likely to progress to recumbency with death resulting from respiratory failure. In our cases, however, despite the rapid progression of the disease, muscular weakness and reduced tail tone were the only observed clinical signs.

A definitive diagnosis of botulism is achieved by the demonstration of the toxin in the serum of the affected animal. In general, cattle and other livestock are more sensitive than humans to *C. botulinum* toxin (4,8). It may be difficult to identify the toxin, since the concentration of toxin in blood is often too low for detection by mouse inoculation. In such cases, when a definitive diagnosis is lacking, a tentative diagnosis can be made by the epidemic nature of the outbreak, the clinical signs of progressive weakness without loss of sensation, the isolation of the infectious agent in the gastrointestinal contents or the detection of toxin in extracts of fecal material, and the direct or indirect access to a source of potentially contaminated material, such as chicken litter.

When botulism is suspected, the 1st critical therapeutic step is to give polyvalent antitoxin to affected animals. It should be noted that the antitoxin is only effective against the toxin in circulation or bound to a receptor on the external surface of the nerves; it is not effective when the toxin is fixed at the neuromuscular junction (9). Antibiotics are only indicated when there is a suspicion of inhalation pneumonia or wound infection. Some authors suggest avoiding the utilization of antibiotics that may potentiate neuromuscular weakness; for example, aminoglycosides, tetracyclines, and procaine penicillin (1). Other therapeutic procedures include supportive care (oral water and electrolytes) and reduced physical activity.

One author (4) reports that the prognosis is worst when the disease progresses rapidly and the animal is recumbent, in which case most adult cattle will die. Considering the rapidity of the onset of severe clinical signs and the absence of antitoxin therapy, the survival of the 3rd steer is surprising. Recovery from botulism occurs by regeneration of new nerve endings, not by metabolism or elimination of the toxin (10).

One author reported that recovery from botulism does not result in the development of immunity (4). In the present case, it was possible to observe the neutralizing effects of the serum of a surviving steer over a serum of a dying steer using mouse inoculation tests. The

serum of the surviving steer seemed to contain a substance that inhibited the action of the toxin presumed to be present in the serum of the dying steer and suggested the development of immunity by the 3rd steer.

Since poultry are most often affected by type C toxin (occasionally by types A and E) (10), and cattle are susceptible to types B, C, and D, it is most likely that cattle consuming poultry litter will be affected by type C toxin. However, cattle commonly develop *C. botulism* type B in the United States (4).

The question arises as to whether the toxin is present in an active form in the muscle of a clinically affected animal. One study demonstrated the presence of type C toxin in the muscle tissue of clinically affected cattle (11). Type C toxin is reported to be denatured by heating to a temperature of 90°C for 2 min. Although most authors agree that type C toxin does not cause human disease, animals suffering from botulism are not considered fit for human consumption and should not be presented for slaughter. In the use of feed, such as ensiled poultry litter, it is essential that the silage is made carefully and free from poultry carcasses. In addition, vaccination (Toxoid C/D, Commonwealth Serum Laboratory, Parkville, Victoria, Australia), which is an effective method of control in endemic areas, is strongly recommended when there is access to potential toxin-containing substances.

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References

1. George LW. Diseases of the nervous system. In: Smith BP, ed. Large Animal Internal Medicine. St-Louis: CV Mosby, 1990: 1033–1035.
2. Radostits OM, Blood DC, Guay CC. Veterinary Medicine, 8th ed. Philadelphia: Baillière Tindall, 1994: 680–683.
3. Trueman KF, Bock RE, Thomas RJ, *et al.* Suspected botulism in three intensively managed Australian cattle herds. Vet Rec 1992; 130: 398–400.
4. Whitlock RH. Botulism in cattle. 6th Annu Symp Adv Clin Vet Med, University of California 1993: 64–71.
5. Haagsma J, Ter Laak EA. Atypical cases of type B botulism in cattle caused by supplementary feeding of brewer's grains. Neth J Vet Sci 1978; 103: 312–325.
6. Divers TJ, Bartholomew RC, Messick JB, *et al. Clostridium botulinum* type B toxicosis in a herd of cattle and a group of mules. J Am Vet Med Assoc 1986; 188: 382–386.
7. Gray TC, Bulgin MS. Botulism in an Oregon dairy cow herd. J Am Vet Med Assoc 1982; 180: 160–162.
8. Whitlock RH. Botulism in Large Animals. Proc 8th Forum Am Coll Vet Int Med, Washington, DC. 1990: 681–684.
9. Simpson LL. The origin, structure and pharmacological activity of botulinum toxin. Pharmacol Rev 1981; 33: 155–185.
10. Duchen LW. Motor nerve growth induced by botulinum toxin as a regenerative phenomenon. Proc R Soc Med 1972; 65: 196–197.
11. Neill SD, McLoughlin MF, McIlroy SG. Type C botulism in cattle being fed ensiled poultry litter. Vet Rec 1989; 27: 558–560.